

1 and 2. Using the LCModel postprocessing software (S. Provencher Inc., Ontario, Canada), metabolite ratios were compared with data from 11 healthy volunteers.

**Results.** Hyperintensity of the cerebellar cortex on T2-weighted and fluid-attenuated inversion recovery images was the most striking finding on MRI. Cerebellar foliation was normal. Cerebellar tissue was reduced with widening of the cerebellar fissures, most pronounced in the vermis, and enlargement of the fourth ventricle. The brainstem and the size of the posterior fossa appeared normal. No white matter abnormalities were observed, and myelination was normal for age. The basal ganglia, cerebral cortex, and pituitary gland were also normal (figure). MRI changes in Patient 2 were not progressive.

MRS revealed reduced *N*-acetylaspartate/Cr (mean  $\pm$  SD, 0.36 and 0.49 vs  $1.36 \pm 0.18$  in controls) and increased myo-inositol/Cr (1.46 and 1.47 vs  $0.72 \pm 0.15$ ) metabolite ratios in the cerebellum. In Patient 2, myo-inositol/Cr was also elevated in the parieto-occipital white matter ( $1.13$  vs  $0.65 \pm 0.09$ ). Basal ganglia spectra were normal.

**Discussion.** Although hyperintense cerebellar cortex in combination with extensive white matter changes was recently described in a patient with a mitochondrial disorder,<sup>3</sup> hyperintensity of the cerebellar cortex is the neuroradiologic hallmark of INAD and considered pathognomonic for this disorder.<sup>1</sup> INAD is a rare autosomal-recessive disorder involving CNS fiber tracts and peripheral axons. Clinically, it is characterized by infantile onset and rapid progression of psychomotor regression and hypotonia, evolving spasticity, and visual disturbance caused by optic atrophy.

In contrast, our patients had nonprogressive psychomotor retardation, ataxia, and cataracts, suggesting the diagnosis of MSS. Other frequent features of MSS include muscle hypotonia, muscle weakness and atrophy, and a short stature. Hypogonadism observed in some patients has been correlated with a small or absent pituitary gland on MRI. Skeletal anomalies with scoliosis and chest and foot deformities have also been reported. Diagnosis is based on typical clinical findings, the presence of autophagic vacuoles, and unique dense membranous structures associated with cell nuclei in biopsied muscle.<sup>2</sup> These were all demonstrated in our patients.

Cerebellar changes, more pronounced in the vermis than the hemispheres, are the most common imaging finding in MSS.<sup>4,7</sup> Variably described as hypoplasia or atrophy, they consist of either a small, compact cerebellum without widened fissures<sup>6</sup> or a normally sized cerebellum with wide fissures and an enlarged fourth ventricle.<sup>4,6,7</sup> Additional findings are absence of the posterior bright spot and/or a small anterior pituitary gland,<sup>6</sup> supratentorial

white matter abnormalities, and cerebral atrophy, the latter more commonly observed in adolescent and adult patients.<sup>5,6</sup>

Apart from the additional finding of a distinctly hyperintense cerebellar cortex, the cerebellar changes in our patients were consistent with the findings reported in MSS, namely, nonprogressively widened cerebellar fissures and an enlarged fourth ventricle within a normal bony posterior fossa.

Interestingly, on histopathology there is severe atrophy of the cerebellar cortex with neuronal loss and astrogliosis in MSS and INAD. This similarity of underlying histopathologic changes may explain the occurrence of a T2-hyperintense cerebellar cortex in the two diseases.

In conclusion, hyperintensity of the cerebellar cortex on T2-weighted images is not pathognomonic for INAD but also can be a characteristic finding of MSS.

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## Dental diplopia with transient abducens palsy

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Diplopia is uncommon after intraoral local anesthesia for dental procedures.<sup>1-7</sup>

**Case report.** A healthy 29-year-old man was examined at a hospital-based dental clinic several days after sustaining mild facial trauma during a snow skiing accident. He had dental pain and thermal sensitivity, which related to fractured cusps of the bottom right second premolar tooth (no. 29). Because of the severity of the cusp fracture, the treatment plan was to remove this tooth. The patient's medical history was otherwise unremarkable, and he denied any allergies. The dentist proceeded to anesthetize the patient with a right inferior alveolar nerve block using a standard 25-gauge needle (1.5 inches) and 1.8 mL of 2% lidocaine with 1:100,000 epinephrine. No other anesthetic or analgesic was used during this procedure. Initial aspiration of the needle was devoid of blood, but the patient did note a brief twinge in his right lower lip at the time of the injection. Within 5 minutes of the injection, profound anesthesia was obtained in the expected distribution of the inferior alveolar nerve. Shortly thereafter and before the procedure began, the patient had new-onset diplopia with rightward gaze. The dentist performing the procedure observed disconjugate eye movements and transferred the patient to the emergency department. Further history revealed that the patient had previously undergone multiple dental procedures without

complication and had no previous adverse reactions to anesthetics or other medications. The general examination was unremarkable, and neurologic examination was abnormal only for numbness in the distribution of the inferior alveolar nerve, esotropia, and inability to fully abduct the right eye. The remainder of extraocular movements and other cranial nerves were normal, and Horner syndrome was not present. Within ~1 hour of onset, the numbness and diplopia resolved, and the patient returned to his premonitory state.

**Discussion.** Ophthalmoplegia after intraoral, local dental anesthesia has been well described in the dental literature<sup>1-7</sup> and commonly manifests as transient abducens palsy as in our patient. The mechanism of the ophthalmoplegia is uncertain, although the route through the pterygopalatine fossa is likely because it is the only vascular-rich structure between the injection site and the abducens nerve. Extravascular spread of the anesthetic could have occurred along a dural plane anywhere near the injection site. The anesthetic could have diffused across the pterygopalatine fossa and reached the laterally located abducens nerve via the anterolateral extension of the inferior orbital fissure. Involvement of other cranial nerves serving eye movements and pupils would be more difficult to explain with this mechanism than the others.

Intravascular injection could have occurred despite careful aspiration before the administration of anesthetic. Aberrant arterial patterns and retrograde flow have been suggested to explain how the anesthetic could access structures necessary to produce ophthalmoplegia after an intra-arterial injection. Perhaps more

likely, the anesthetic could have gained access to nervous system structures in the cavernous sinus because of IV injection or absorption. As has been proposed for some infections, local anesthetic could have drained into the pterygoid venous plexus and thereby into the cavernous sinus via emissary veins traversing bony foramina, especially when the patient is in the recumbent position. Here the abducens nerve may be more susceptible than other cranial nerves because it courses through the cavernous sinus rather than in its wall, like other cranial nerves. Features of Horner syndrome seen in some patients<sup>7</sup> could also be explained by the anesthetic affecting sympathetic fibers that accompany the carotid artery through the cavernous sinus.

Fortunately, the anesthetic effects of lidocaine are short lived, and permanent abducens palsy has not been reported in this setting. Management is usually supportive, but a protracted course, as previously described,<sup>4</sup> should prompt neuro-ophthalmologic evaluation. Undaunted by his episode of diplopia, the patient returned to the dental clinic later the same day to resume treatment, which involved repeat inferior alveolar nerve block. No further incidents occurred at the time of treatment or on follow-up evaluation.

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## Postpartum obturator neuropathy

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**Case report.** A previously well 34-year-old woman sought treatment postpartum for left leg weakness. Several hours after labor onset, pain was noticed in the left groin and medial thigh. Epidural anesthesia was commenced via the L3/L4 interspace. The pain resolved within 15 minutes. A deep transverse arrest occurred, and semi-urgent cesarean section was performed without complication. Difficulty in left leg adduction was apparent the following day with walking requiring assistance. Bladder, bowel, and contralateral leg function were normal.

Examination revealed profound weakness of left thigh adduction only. Knee and ankle reflexes were normal bilaterally with flexor plantar responses. The left adductor reflex was significantly reduced, whereas the right was easily elicited. A small area of decreased pinprick sensation was present over the medial left thigh. MRI of the lumbosacral spine was performed 5 days postpartum with no evidence of lumbar nerve root compression.

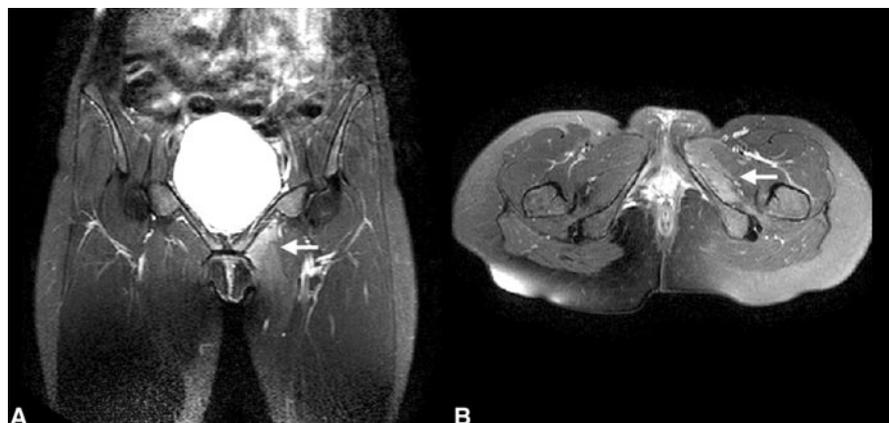
Three weeks later, significant weakness of left thigh adduction remained. The adductor reflex was more symmetric, and sensation was normal. Routine nerve conduction studies were normal. EMG revealed active denervation in the left adductor magnus with no motor units under voluntary activation. Other muscles innervated by the left L2 to L4 nerve roots were normal. MRI of the pelvis and thigh demonstrated increased signal on T2-weighted images in the adductor brevis, obturator externus, and adductor magnus muscles, consistent with denervation (figure).

No focal compressive lesion or signal change within the obturator nerve was seen.

**Discussion.** The obturator nerve is formed by fibers from the ventral divisions of the second, third, and fourth lumbar nerves.<sup>1</sup> As the nerve crosses the upper margin of the obturator internus muscle, it is vulnerable to compression against the lateral wall of the pelvis. The nerve then curves downward and forward around the lateral wall to traverse the obturator foramen. Within this foramen, the nerve divides into anterior and posterior branches. The anterior division innervates the adductor longus, gracilis, and adductor brevis muscles and gives off sensory branches to the medial thigh. The posterior division innervates obturator externus and adductor magnus.

Obturator neuropathy causes weakness of thigh adduction as the only motor manifestation because other nonobturator-innervated muscles participate in lateral hip rotation. Medial thigh pain can occur and may radiate toward the knee. Altered sensation typically occurs over the medial aspect of the mid and lower thigh. Complete loss of sensation is unusual because of overlap from neighboring cutaneous nerves, and occasionally there is no sensory loss. In chronic cases, wasting of the medial thigh is seen. During ambulation, the hip is abnormally abducted, resulting in a circumducting, wide-based gait. Ipsilateral loss of the hip adductor tendon reflex can suggest obturator neuropathy; however, because it is sometimes absent in healthy people, the contralateral reflex must be easily elicited for the finding to be useful.

Obturator nerve lesions are uncommon, primarily because of its protected location deep within the pelvis and medial thigh.



*Figure. Coronal (A) and axial (B) fat-suppressed T2-weighted MRIs of the upper thigh showing increased signal within the adductor brevis and adductor magnus on the left (arrows). Atrophy of these muscles is also evident.*